Bull, (C.S.)

A CONSIDERATION

OF

SOME UNUSUAL FORMS OF INTRA-OCULAR HEMORRHAGE, WITH SPECIAL REFERENCE TO ETIOLOGY AND PROGNOSIS

BY

CHARLES STEDMAN BULL, A.M., M.D.

Surgeon to the New York Eye and Ear Infirmary; Consulting Ophthalmic Surgeon to St. Mary's Free Hospital for Children, and to the Nursery and Child's Hospital, New York

Reprinted from THE MEDICAL RECORD, *December* 4, 1886

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CONSIDERATION OF SOME UNUSUAL FORMS OF INTRA OCULAR HEMORRHAGE, WITH SPECIAL REFERENCE TO ETIOLOGY AND PROGNOSIS.

The class of cases to which I desire to call attention has interested me for some years, both because of their rarity and because of their somewhat doubtful causation. I refer to those cases of intra-ocular hemorrhage occurring in persons of middle and advanced life, who have been subjected to severe malarial poisoning. In these cases the blood is extravasated into the vitreous humor or aqueous humor, or both, and the attack is entirely unaccompanied by any other signs of intra-ocular inflammation. The hemorrhages always occur suddenly, and usually into the posterior part of the vitreous humor. The sudden and sometimes extensive loss of sight, together with a direct examination of the media of the eye, in these cases enable us to diagnose an intra-ocular hemorrhage as the cause of the disturbance. Very little has been written in regard to the effects of malarial fevers upon the internal tunics of the eye, though severe lesions of the optic nerve, with amblyopia and amaurosis, are now generally recognized as being occasionally produced by prolonged exposure to miasmatic influences. The occurrence of intra-ocular hemorrhages is merely hinted at, and yet when we consider the not infrequent occurrence of hemorrhages in other parts of the body in persons who have been the victims of protracted pernicious malarial poisoning, it should not surprise us that hemorrhages from the same cause may occur within the eye. Intestinal hemorrhage, epistaxis, bleeding from the gums, hæmaturia, and other signs of a general hemorrhagic ten-

dency have been noted in persons suffering from severe miasmatic poison, especially in tropical countries, and in the southwestern regions of our own country, where the malarial fevers are so often of the congestive type. As a result of venous congestion caused by rapidly occurring temporary obstructions in the capillary circulation, it is known that small, even punctate, hemorrhages sometimes occur in the vitreous entirely independently of pulmonary or cardiac disease. When such hemorrhages are not due to inflammatory changes in the retina or choroid, Wecker and other authorities think that they are probably caused by some disturbance in the circulation, such as venous stasis or increase in the arterial tension, or else by disease of the vascular walls, such as fatty or atheromatous degeneration. Very often several of these causes combine to produce the hemorrhage. Hypertrophy of the left ventricle is now recognized as an important factor in the causation of retinal apoplexy, and presumably, also, in certain cases of hemorrhage into the vitreous. Fatty degeneration is also sometimes indicated by these vitreous hemorrhages. Another possible cause which of late has been brought forward is a gradual dilatation and hypertrophy of the entire vascular system—cardiac and vascular dilatation, without valvular disease or general cyanosis. We also admit that changes in the character of the blood, such as exist in progressive pernicious anæmia, sometimes cause these retinal hemorrhages. It is my belief that diminished intra-ocular tension, or any considerable change in tension, may give rise to vitreous hemorrhages. In such cases pulsation in the retinal arteries is frequently isochronous with the diastole of the radial artery, and this pulsation may be followed far beyond the margin of the optic disk. These pulsations in the retinal arteries occur almost constantly with insufficiency of the aortic valves, and yet may at times be entirely absent. The reason for this is that the change of tension in the arterial system is much greater than in a healthy circulatory sys-

tem. It was formerly almost universally considered that large extravasations of blood into the vitreous came from ruptured choroidal or ciliary vessels ; but some observers have come now to believe that in these cases the blood comes either from the papillary and retinal vessels, which are branches of the central retinal artery, or from the vessels of the sheath of the optic nerve. This is the view advanced by Wecker, and it has many adherents. Wecker states that in very many cases the blood may be traced directly to the papillary margin, or to some one branch of the central retinal artery. These "sheath-apoplexies," as he calls them, may occur successively in the two eyes, and are not infrequently met with in diabetic patients. Where they occur along the course of the larger vessels for some distance, it is sometimes difficult to distinguish them from retinal apoplexies.

It is certainly important to carefully observe the relation of these vitreous hemorrhages with the margin of the optic disk, and as soon after their extravasation as possible, which, of course, can only be done when the hemorrhage is a slight one. Wecker is of the opinion that they occur after a sudden change of temperature, or after some ordinary muscular exertion made by persons when in a state of congestion or fever, and are fifty years of age or over, or who are suffering from cardiac disease. It is probable, also, that similar hemorrhages occurring in young people are produced in the same way ; but as the retina is more resistant in them and less liable to tear, the extravasated blood is more apt to pass forward in the retina, or between the latter and the vitreous membrane, and enter the vitreous humor in its anterior segment just behind the lens. Small extravasations of blood in the anterior portion of the vitreous sometimes occur after violent muscular efforts or exposure to malarial disease, but they are very different from these sudden and large extravasations.

A general filamentous or flocculent opacity of the vitreous is a not very infrequent result of one of these

hemorrhages. These opacities may or may not be adherent to the retina and choroid, and when the pupil is well dilated they are seen to be suspended in the vitreous. These may exist without any other lesion of the eye, and might possibly be regarded as the consequence of periodic congestion of the blood-vessels with rupture and extravasation of blood into the vitreous.

As before stated, it is Wecker's opinion that most of these hemorrhages come from the vessels of the sheath of the optic nerve, and its method of occurrence he describes as follows: "The blood here at first opens a passage through the natural openings, follows the eccentric course of the hyaloid canal, and thence enters into the 'triangular space,' so called, of Stilling." Wecker considers that what Manz has called "retinitis proliferans" is nothing but the organization of large strata or layers of blood extravasated in the posterior part of the vitreous. These layers become in time thinner and more translucent, and in contracting cause a deviation of the central vessels toward the point of implantation of these membranes. This view will, however, scarcely meet with general acceptance. He claims to have seen patients who have become suddenly blind from intra-ocular hemorrhage, in whose eyes the vitreous was occupied by several translucent membranes which divided it into regular segments. These, he asserts, are formed at first by extravasations of blood from the margin of the papilla which project into the vitreous, and says that he has in several cases been able to follow the various stages of their development and transformation.

These hemorrhages, assumed to be due to malarial poisoning, occur almost always in one eye, though they may occur in both eyes; but I have never seen a case in which they occurred simultaneously in both eyes. In every instance in my experience they have happened during the febrile or congestive stage. All my patients were forty years of age or over, and had been subject to malarial disease for a varying length of time, contracted

in markedly miasmatic regions. In some the tension of the eye was increased ; in others it did not seem to have suffered any change. The refraction was in the majority of cases slightly hypermetropic, while in a smaller number it was emmetropic, and in a few cases it was slightly myopic. In the majority of cases the blood seemed to be extravasated generally through the vitreous, but in a few cases the mass of the blood was certainly in the posterior part of the vitreous. The recurrences of the hemorrhages were always, as in the first instance, spontaneous, and sometimes frequent. The process of absorption was always very slow, even when but one hemorrhage occurred ; and in some instances, after a brief period, the absorption seemed to be entirely stayed. In those cases in which the blood disappeared sufficiently to admit of a more or less satisfactory view of the fundus, there was never any arterial or venous pulse. In no case was there any valvular disease of the heart ; but in some of the cases there was hypertrophy of the left side of the heart, though never to a marked degree. In a few cases there was an atheromatous condition of the arteries, as in the temporal and radial vessels, and in all of them there was a more or less marked irregularity in the strength and quantity of the blood-current. In no case was there complete restoration of central vision. In nearly all the cases there existed peripheral choroidal disease, usually atrophic in character and slight in degree, which could be distinguished with the ophthalmoscope after the hemorrhage had been partially absorbed. In none of the cases was there any tendency to either gout or rheumatism, such as Hutchinson describes in cases of spontaneous vitreous hemorrhage. In his cases there were always iritis, with vitreous opacities as well as hemorrhages, and he thought that the tendency to extravasation of blood was due to unequal circulation. We know from recent investigations that both low tension and high tension are compatible with liability to rupture of the capillaries. A condition of loss of balance

is easily induced if the blood-vessels are not well under vaso-motor control, and they readily become empty in one place and overfull in another. In all my cases chronic renal disease was excluded by a careful and frequently repeated examination of the urine. There seems no reasonable doubt that the hemorrhages were due in the main to rupture of the choroidal or ciliary vessels, and possibly of the vessels of the sheath of the optic nerve, during the febrile or congestive stage of malarial attack, when there was a loss of balance in the vascular tension, and when the system had become decidedly enfeebled by previous attacks of a similar nature.

As regards the *prognosis* of these cases, it of course depends largely on the cause, the amount of blood extravasated, and the degree of organization to which the resulting opacities may have attained. If the choroid and ciliary body were in a healthy condition, these hemorrhages might be absorbed more rapidly and completely provided the patient could be removed from his miasmatic surroundings and a proper treatment and diet instituted. But unfortunately in these cases there was more or less atrophy of the uveal tract, and the blood disappeared very slowly. Any obstacle to the proper nutrition of the vitreous would of course stand in the way of the absorption of these hemorrhages, and this, of course, is always the case in large and sudden extravasations of blood. The most that can be hoped for in these cases is that they will eventually become such thin membranes as not only to be semi-transparent, but will also cease, by their contraction, to pull on the retina, and thus cause its possible detachment. These membranous opacities, the remains of organized hemorrhages, may and do last for years.

Case I.—C. H——, aged forty, travelling salesman; first seen March 27, 1874. Three years before, while in Texas, he was dangerously ill with quotidian intermittent fever of the congestive type, and on recovering con-

sciousness, after a second attack, he noticed he could not see with the *right* eye. For six weeks the sight of the eye was so defective as to be useless, and he then began slowly to regain his vision. It was more than a year before he could see to read, and since then there have always been floating particles and thread-like bodies before this eye. He has had several attacks of fever since then, and with each one a more or less marked obscuration of vision in the right eye. During the last week in February, 1874, while under treatment, and walking in the street, he had a slight chill, which was very soon followed by high fever, and while in the febrile stage the sight of the *left* eye began to fail, and in fifteen minutes he was entirely blind. When I saw him the examination revealed: R. E., $\frac{2}{3}$. L. E., perception of light; tension + 1. Several filiform and membraniform opacities in right vitreous, and peripheral choroidal atrophy. In left eye, dull reflex from fundus at periphery, and vitreous full of blood. No cardiac or renal disease. Liver enlarged. Refraction hypermetropic, D. 1.50.

Case II.—P. K.——, aged sixty-eight, farmer; first seen August 10, 1874. When a young man he had intermittent fever of the ordinary tertian type, while living in New Jersey, which obstinately resisted treatment until a sea-voyage of some duration broke it up. Subsequently he settled in Indiana, and during his residence there on a farm became subject to severe chills and fever. Has had a number of attacks of loss of sight in both eyes, but does not remember how long they lasted. Thinks his vision much impaired in *left* eye. One week ago vision in *right* eye began to fail very rapidly, while suffering from the second stage of an attack of fever. Examination showed: R. E., $\frac{1}{20}$; tension — 1; blood in anterior chamber; large hemorrhage in vitreous; peripheral choroidal atrophy. L. E., $\frac{2}{20}$; extensive peripheral choroidal atrophy; two small membranes extending diagonally across vitreous in the axis of vision, apparently attached downward and backward to retina on temporal

side; small recent hemorrhage in vitreous, just above and behind the lens on nasal side; tension normal; myopia, D. 1. Hypertrophy of left side of heart. Atheroma of arteries at wrist and temple. No renal disease. This patient was under observation only for a brief period of three months, during which time there was scarcely any improvement of vision and no change in fundus.

Case III.—L. S——, aged forty-five; first seen June 25, 1878. Patient resident for many years in Central America, though of German birth. Repeated attacks of congestive fever, and three attacks of serious loss of sight in *left* eye, with only partial restoration of vision. Eight days before I saw the patient he had rather a violent febrile attack, with sudden and complete loss of sight in the *left* eye, but no pain. This total blindness in *left* eye lasted three days, and then slow improvement set in. Examination showed: R. E., $\frac{20}{20}$; media clear and fundus healthy except at periphery, where there were signs of beginning choroidal atrophy. L. E., movements of the hand; tension + 1; in vitreous one large clot and several floating smaller clots. Emmetropia R. E. No cardiac or renal disease.

Vision in this case improved very slowly. At the end of ten months it was $\frac{20}{60}$, and at the end of two years $\frac{20}{10^{0}6}$. The vitreous of the L. E. showed a large floating membrane, which was thicker in some places than in others, and was apparently attached both at the equatorial region and at the optic disk.

Case IV.—S. M. L——, aged forty-six, native of Arkansas; first seen July 17, 1878. Has been subject all her life to congestive chills, but never had any failure of vision till eight months ago, when she suddenly lost the sight of the *left* eye while in one of her attacks. From this she slowly recovered. Four weeks ago, while suffering from an attack of fever, she again lost the sight of *left* eye entirely, and of the *right* eye partially. The *left* eye remained entirely blind for six days, and then vision began slowly

to return. An examination showed: R. E., $\frac{20}{200}$; small recent hemorrhage in the vitreous near the retina, and exactly in axis of vision; refraction hypermetropic. L. E., fingers at 6″ eccentrically; large clot in vitreous obscuring entire fundus except at equator. No cardiac or renal lesion. Liver and spleen both enlarged. Choroidal atrophy at periphery. This patient has never had any recurrence of the hemorrhages, and when last seen vision was, in R. E. $\frac{20}{30}$; L. E., $\frac{2}{200}$, with a somewhat thick membranous band stretching across the centre of the vitreous, which was perforated by several holes, through which the fundus was visible.

Case V.—C. K——, aged forty-three, steward of steamer; first seen February 14, 1879. For the last twelve years has been subject to congestive chills, which he attributes to an attack of yellow fever which he contracted at Vera Cruz. Has never had any trouble with his vision till ten days ago, when, after a violent chill and equally violent fever, he noticed an obscuration of vision of *left* eye which rapidly grew worse, and on the next morning he could not even see light. Vision began to improve on the sixth day. Examination showed: R. E., $\frac{20}{20}$; media and fundus normal; refraction hypermetropic. L. E., movements of hand; vitreous filled with floating blood-clots. No cardiac or renal disease, but spleen much enlarged.

This patient in the course of seven months had four hemorrhages into the vitreous of the L. E. He was then free from attacks of fever for nearly two years, during which vision in L. E. rose to $\frac{20}{70}$. Then came another hemorrhage entirely filling the vitreous, and when last seen, now some years ago, vision had sunk to $\frac{5}{200}$ in L. E.

Case VI.—A. M——, aged forty-five, sailor; first seen December 3, 1879. Has had several severe attacks of congestive intermittent fever while in tropical climates, the last being about eight months ago, while on the west coast of Africa. During this last attack he lost the sight of the *right* eye entirely. The loss of sight was sudden and complete within a few minutes. The eye

also became bloodshot, and remained so for some weeks. Two weeks ago, while on shipboard, he had another chill followed by high fever, and again lost the sight of the *right* eye, which had partially recovered from the previous attack. Examination showed: R. E., $\frac{1}{200}$; tension normal; no reflex from the fundus except at the extreme periphery; small hemorrhage in the anterior chamber, and the vitreous full of blood. L. E., $\frac{20}{20}$; media and fundus normal; refraction hypermetropic. No cardiac or renal disease, but the man's appearance and complexion is that of one saturated with miasmatic poisoning.

In this case vision in the R. E. improved to $\frac{20}{100}$ in fourteen months, but since then there has been no improvement.

Case VII.—B. M——, aged fifty, civil engineer; first seen August 9, 1880. Was healthy up to four years ago, when he contracted congestive chills while in Mexico. For more than a year was unable to do any work. Two months ago, during an attack, vision suddenly failed in the *right* eye, and in about an hour he had only perception of light. Examination showed: R. E., $\frac{20}{100}$; tension normal; numerous small floating clots in the vitreous; choroidal atrophy at the periphery. L. E., $\frac{20}{30}$; faint opaque striæ at periphery of lens; peripheral choroidal atrophy; refraction emmetropic. No cardiac disease and no renal disease, but liver and spleen somewhat hypertrophied.

June 17, 1881.—Sudden and extensive hemorrhage into the vitreous of *right* eye four days ago, which now seems limited to the nasal half of fundus. V. = $\frac{1}{200}$ eccentrically. This case is one of the very few I have seen in which there has apparently never been any absorption of the extravasated blood. The patient was under observation at very long intervals for nearly five years, and at the last visit the vision of the R. E. was $\frac{1}{200}$, and the blood in the vitreous did not show any change.

Case VIII.—B. G——, aged forty-three. First seen October 4, 1880. First attack of fever ten years ago,

and has never been free from it since. First failure of sight in *left* eye seven months ago, from which she partially recovered. Another attack, accompanied by loss of sight, three months ago, and again a sudden and complete loss of sight eight days ago, from which, however, she has already begun to recover. Examination shows: R. E., $\frac{20}{20}$; media and fundus normal; refraction emmetropic. L. E., $\frac{1}{200}$; blood in the anterior chamber and a large clot in the vitreous. Patient emaciated and very feeble. Heart-sounds faint and circulation very sluggish, but no murmurs and no hypertrophy. Enlarged spleen. No renal disease.

This patient has been seen at long intervals since—the last time in May, 1886. An examination then showed: R. E., $\frac{20}{20}$, with some peripheral choroidal atrophy. L. E., $\frac{1}{200}$; large diaphanous membrane in vitreous; the remains of the old blood-clot, and some floating opacities, still tinged with blood, the remains of more recent hemorrhages. She still suffers from chills and fever.

Case IX.—M. N——, aged forty-three, clerk; first seen October 25, 1880. Subject to intermittent fever for many years, but has never lost his sight entirely, although he has had attacks of partial failure of vision on several occasions. One week ago had a violent attack of fever, and suddenly noticed a large black spot before the *left* eye, which rapidly grew larger and soon obscured the entire sight. Examination showed: R. E., $\frac{20}{20}$; hazy vitreous, with what was apparently a small blood-clot. L. E., $\frac{1}{200}$ eccentrically, and vitreous filled with blood; faint reflex at extreme periphery. No cardiac or renal disease.

This patient was under observation daily for about three weeks, and then at long intervals for nearly two years, since which time he has not been seen. During the two years he had several slight attacks of loss of sight in L. E. At date of last visit examination showed: R. E., $\frac{20}{20}$. L. E., $\frac{1}{200}$, with both fixed and floating opacities in the vitreous, and a general haziness which obscured most of the fundus.

CASE X.—H. G——, aged sixty-four, captain of a sailing vessel; first seen in February 25, 1882. Has had several attacks of Chagres fever and one of yellow fever, and in all of them had loss of vision, though never complete. Thinks he has never regained the sight of the *right* eye entirely since the attack of Chagres fever nine years ago. Ten days ago, while at sea and recovering from a chill, there was sudden and complete loss of vision in the right eye. Examination showed: R. E., movements of the hand; fundus absolutely invisible. L. E., $\frac{20}{40}$; Ht. D. 1; considerable atrophy of choroid at periphery; two semi-transparent delicate membranes in the vitreous, the remains probably of former hemorrhages; faint striæ at periphery of lens.

No renal disease, but has hypertrophy of left side of heart. This patient was under observation for about five months, during which time there was no apparent absorption of the blood in the vitreous of R. E. He was again seen in April, 1884, and then vision in R. E. had risen to $\frac{20}{200}$, with some floating opacities and one large membraniform opacity attached to disk.

CASE XI.—J. F——, aged fifty-eight, discharged soldier; first seen February 12, 1883. Contracted fever first while in the army during the Civil War, along the Red River. Subsequently was transferred to the Army of the Potomac, and suffered severely in the swamps of the Chickahominy region. Has never lost the vision of either eye entirely, but has had repeated attacks of partial failure of sight, always during a febrile attack. Has had no distinct attack of fever for several years, though he has had chilly feelings. Three days ago, while complaining of a congested feeling in his head, he suddenly and entirely lost the sight of the *left* eye. Examination showed: R. E., $\frac{20}{30}$+; hypermetropic; choroidal atrophy. L. E., perception of light; vitreous filled with blood; no reflex; tension + 1.

Patient has had muscular rheumatism, but has no organic heart disease. Repeated examinations of the urine show a faint trace of albumen but no casts.

Patient last seen in July, 1885. He had had two attacks of hemorrhage into the vitreous since his first visit to me, in both of which I saw him. Vision at last visit: R. E., $\frac{20}{30}$. L. E., $\frac{15}{200}$; clots and membrane in vitreous.

Case XII.—B. G——, aged forty-four; first seen March 26, 1883. Subject to repeated attacks of intermittent fever of the regular tertian type for many years. First loss of sight two years ago, in *left* eye, during an attack of fever. Second attack about ten months ago in same eye. Third attack two days ago in same eye. Examination showed: R. E., $\frac{20}{20}$; media clear; fundus normal; refraction emmetropic. L. E., $\frac{1}{200}$ eccentrically, eye slightly divergent; fresh hemorrhage into vitreous, and membranous remains of former hemorrhages. Patient thinks that the divergence of L. E. came on after the second attack of loss of sight ten months ago. This patient is in fairly good condition, in spite of the long continuance of the malarial poison in his system. No cardiac or renal disease. Slight tendency to chronic diarrhœa. No choroidal atrophy. This patient was last seen eight months ago, two years after the last hemorrhage, and the absorption had proceeded so slowly that in L. E. V. = $\frac{1}{200}$, and the details of the fundus were still so indistinct that no exact idea of the condition of the choroid and retina could be obtained.

Case XIII.—P. McB——, aged forty, carpenter; first seen April 16, 1883. Contracted intermittent fever of a virulent type three years ago, and with the third attack had loss of sight in the *left* eye, from which he says he entirely recovered. After that he had repeated attacks in the same eye, generally slight, and always during or after a febrile attack. The last attack occurred three weeks ago, and was the most severe he had ever had.

Examination shows: R. E., $\frac{20}{30}$. L. E., $\frac{2}{200}$. Media of R. E. clear, but in the L. E. a large floating clot and numerous thin, almost transparent filaments, the remains probably of former hemorrhages. Refraction hypermetropic. No cardiac or renal disease. No apparent en-

largement of liver or spleen. Vigorous treatment, persisted in for about four months, succeeded in curing this case as far as the febrile attacks were concerned, and there was no recurrence of the hemorrhage into the vitreous. This patient was seen during the past summer, but vision in the L. E. had only improved to $\frac{15}{200}$, with no distinct view of the fundus.

Case XIV.—R. S——, aged fifty-six; first seen December 31, 1883. Subject to "congestive chills" for some years, contracted in southwestern Missouri. Has had repeated attacks of partial and transient loss of sight, lasting for a few weeks. Sudden failure of vision in right eye two days ago. Has rather marked yellow complexion, and is very thin.

Examination shows; R. E., $\frac{20}{200}$: faint reflex from fundus; large clot in vitreous. L. E., $\frac{20}{15}$; choroidal atrophy at periphery; small membranous opacities in vitreous. Faint striæ at periphery of both lenses. Refraction, myopic. No cardiac or renal disease. Spleen enlarged. General nutrition depraved. This patient did not recover from the attacks of intermittent fever till the spring of 1885. Between December, 31, 1883, and March 20, 1885, there occurred two hemorrhages into the vitreous. The patient was last seen in November, 1885, when the vision of R. E. was $\frac{20}{200}$, and that of the L. E. was unchanged. The absorption of the blood was very slow.

Case XV.—M. E——, aged forty-seven; first seen November 16, 1885. Always in feeble health, neuralgic, and dyspeptic, but had never had any symptoms of malarial poisoning until the preceding August, while in a low, swampy region. She was exposed one evening to a wetting, and that night had a violent chill, followed by high fever. The next afternoon the chill was repeated and during the febrile paroxysm she suddenly lost the sight of the *left* eye, and has only recently begun to regain it. Examination showed: R. E., $\frac{20}{15}$; faint opacities at lens peripherally; ash. of D. 1; axis 90°. L. E., per-

ception of light; faint reflex from equatorial region; vitreous filled with blood. This patient has no cardiac or renal disease, and no demonstrable engorgement of either liver or spleen. The menopause has not yet begun. Radial pulse feeble and irregular; complexion muddy.

This patient was last seen in June, 1886. At that time R. E., $\frac{2}{20}$. L. E., $\frac{1}{200}$; faint signs of choroidal atrophy at periphery of both eyes; absorption of hemorrhage in L. E. proceeding very slowly; fundus entirely indistinct.

Case XVI.—C. R. F——, aged forty; first seen February 1, 1886. Has had repeated transient loss of sight in one or the other eye, always during a febrile paroxysm, so that, as he said, "he had come to expect it." Subject to severe attacks of intermittent fever, in some of which he was unconscious during a period of about three years. The last attack was one week ago, and was not particularly severe, but was followed by a sudden loss of sight more serious than for a long period.

Examination showed: R. E., $\frac{2}{3}$; small, fixed, and floating opacities in vitreous; choroidal atrophy at periphery. L. E., $\frac{2}{200}$; large clot floating in vitreous; choroidal atrophy. Refraction of R. E. hypermetropic. This patient has no cardiac disease or renal disease demonstrable. Has always been subject to "fever and ague," and the spleen seems slightly enlarged. The blood-current seems feeble, and the general nutrition of the patient much below par. This patient was last seen during the latter part of September, and then vision was unchanged. There did not seem to have been any fresh hemorrhage, and no attempt at absorption had taken place.

Case XVII.—L. C. G——, aged fifty-two; first seen May 17, 1886. Patient has just arrived from Panama. Has had Chagres fever twice; the second time with loss of sight in *right* eye, four years ago, from which he has never entirely recovered. Three weeks ago, while in the interior, had a violent attack of fever, during which he entirely lost sight of *left* eye. Had to be carried on

board the vessel sailing for home. During the voyage there was a slight improvement of the vision.

Examination shows: R. E., $\frac{2}{40}$; a small, long, membranous opacity in vitreous, almost diaphanous, and apparently fixed to retina. L. E., fingers eccentrically at two feet; large floating clots in vitreous, and a general haziness, so that no detail of fundus can be seen. Refraction cannot be estimated, probably hypermetropic. Heart and kidneys apparently normal, but the liver, and especially the spleen, are enlarged. This patient is still under observation; vision in R. E. remains the same, but in the L. E. has increased to $\frac{1}{200}$. Very little absorption of the blood-clot in the vitreous.

As regards the *treatment* of these cases there is little to be hoped for owing to the age of the patients, the atrophic choroiditis, and the consequent retardation and incompleteness of the process of absorption. Wecker was the first to recommend the internal administration of an infusion of jaborandi, and subsequently the hypodermatic injection of the salts of pilocarpine, for chronic vitreous opacities, other than hemorrhagic, so frequently met with in inflammations of the uveal tract; and he subsequently advised their employment in the treatment of the remains of hemorrhages into the vitreous. But hypodermatic injections of pilocarpine should always be employed with great care, and the daily dose should be increased very cautiously for fear of the occurrence of collapse from heart-failure. I cannot recall a single case in which the absorptive process was hastened by the use of this remedy in chronic cases, and I have seen some alarming instances of collapse from its employment. I have seen no benefit arise from the use of diuretics or purgatives, even when continued for a long time in small doses largely diluted. The few partially satisfactory results which I have gained in these cases have apparently been due to the strict attention paid to the dietetic and hygienic management of the patient, such as dry massage with a coarse towel or hair gloves twice or

oftener during the day, and the ingestion of food that was easily assimilated; systematic daily exercise in the open air when the weather permitted, and abstinence from all use of the eyes for close work. Whenever there was a recurrence of the hemorrhage, rest in bed for a few days and the application of a pressure-bandage for a few hours daily seemed to be indicated, and perhaps succeeded in checking the extravasation. A pressure-bandage, however, is not borne well by these patients for more than a few hours at a time. Much has been claimed for the continuous current in promoting the absorption of these opacities, but in my experience it has completely failed. Le Fort, Onimus, and Giraud-Teulon have reported some remarkable results; but Onimus also states that an ill-timed employment of the continuous current has been known to increase the opacities of the vitreous. He thinks that the centrifugal current is the only one which possesses the power of clearing up these opacities of the vitreous—that is, where the negative electrode is placed upon the closed lids, and the positive electrode behind the ears or at the nape of the neck; and the current should not be passed for more than two or three minutes, for fear of increasing the turbidity of the vitreous. In not a single one of the cases which have come under my own observation was any improvement noted from this method of treatment, though carefully carried out and in every instance followed by an ophthalmoscopic examination.